to inhibit the biosynthesis of prostaglandins. Preferential inhibition of prostaglandin E synthesis in the lung, permitting the action of prostaglandin $F_{2\alpha}$, a potent bronchoconstrictor, to remain unopposed, would explain the asthmatic response. The mechanism underlying urticaria and angioedema provoked by aspirin could be a different one, although the role of prostaglandins has not been excluded. In many patients with chronic urticaria, aspirin seems to act as a nonspecific potentiator. The results of experiments to study the effect of tartrazine, benzoates and sulfur dioxide on prostaglandin biosynthesis will be eagerly awaited.

Feingold has proposed that many unspecified food additives and "natural salicylates" may cause hyperactivity and learning disabilities in some children. This theory has gained popular support, but several recent controlled studies of his additive- and salicylate-free diet have shown equivocal or no improvement in children with these disorders.

Food additives, numbering in the thousands, are widely employed to alter the color, taste, and texture of the food and to preserve freshness and inhibit contamination. It is therefore not surprising that some patients may exhibit idiosyncrasy or allergy to one or more of these substances.

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REFERENCES

Michaelson G, Juhlin L: Uticaria induced by preservative and dye additives in food and drugs. Brit J Dermat 88:525-532, Jun 1973

Stenius BS, Lemola M: Hypersensitivity to aspirin and tartrazine in patients with asthma. Clin Allergy 6:119-129, Mar 1976

Freedman BJ: Asthma induced by sulfur dioxide, benzoate, and tartrazine contained in orange drinks. Clin Allergy 7:407-415, Sep 1977

Chaffee FH, Settipane GA: Asthma caused by FD&C approved dyes. J Allerg 40:65-72, Aug 1967

The Use of Antihistamines in Bronchial Asthma

ANTIHISTAMINES have been considered to provide either no benefit or even to be contraindicated in all asthmatic patients. However, recent studies have shown that these drugs do not necessarily cause a deterioration of the asthmatic state, and in fact may provide benefit. Antihistamines not only block H_1 receptors and thus partially block allergen induced bronchospasm, but they appear to have an anticholinergic effect as well. Popa has reported that high doses of chlorpheniramine given intravenously result in a significant degree of bronchodilation in asthmatic patients without atropine-like side effects. Furthermore, Karlin and co-workers have shown improved pulmonary function in patients with mild asthma receiving twice the usual recommended dosage. No adverse effects were seen in patients with chronic severe asthma receiving antihistamines for coexisting allergic rhinitis or urticaria.

These recent studies warrant the following conclusions:

- Asthmatic patients with allergic rhinitis may safely receive antihistamines in the usual dosage, and there is no evidence that coexisting asthma will be exacerbated.
- Although antihistamines can improve asthma in some cases, their use in asthmatic patients must be established on an individual basis.
- The use of antihistamines in patients with status asthmaticus cannot be recommended at this time because of the existence of more effective drugs and because potential drying effect of antihistamines on bronchial mucus has not been firmly clarified.

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REFERENCES

Booij-Noord H, Orie NG, Berg WC, et al. Protection tests on bronchial allergen challenge with disodium cromoglycate and thiazinamium. J Allergy 46:1-11, Jul 1970

Popa VT: Bronchodilating activity of an H_1 blocker, chlorpheniramine. J Allergy Clin Immunol 59:54-63, Jan 1977

Karlin JM: The use of antihistamines in allergic disease. Pediatr Clin North Am 22:157-162, Feb 1975

The Measurement of IgE

THE AMERICAN ACADEMY OF ALLERGY has appointed a Committee on Standardization of In Vitro Tests which is charged with defining and correcting the problems in the quantitation of IgE. They conducted a nationwide single-blind evaluation of the accuracy and reproducibility of the quantitation of IgE which showed an unacceptable level of variation. Besides the large variation between laboratories, the variation of repeated assays within laboratories was unacceptably large.

Part of the problem is undoubtedly due to differences in assay methods. A study of the various assay methods—radioimmunosorbent test (RIST), double-antibody radioimmunoassay (RIA), radial immunodiffusion (RID) and paper disc immunosorbent test (PRIST)—shows that RIST and RID may lead to spurious elevations of IgE in sera and secretions. Double-antibody RIA and PRIST provide the best agreement, but PRIST may give deceptively low results in certain sera. When all 14 laboratories assayed the test serum with the PRIST kit, variations within and between laboratories (133 units per ml to 330 units per ml) were still more than 2½ fold. The problems of the quantitation of IgE are being resolved and a "predictive" table of serum IgE levels in infants,

children and adults, such as the one that follows, may be of value.

IgE—units per ml				
Infants	Children and Adults		Percent of the	Allergic
<2 yrs.	>3 yrs. (RIA)	(PRIST)	Population	Percent
<10	<100	<65	68	<5
10-20	100-350	65-230	20	20
21-100	351-750	231-500	9	35
>100	>750	>500	<3	~100*

*In the absence of helminthiasis.

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REFERENCES

Bazaral M, Hamburger RN: Standardization and stability of immunoglobulin E (IgE). J Allergy Clin Immunol 49:189-191, Mar 1972

Gleich GJ, Dunnette SL: Comparison of procedures for measurement of IgE protein in serum and secretions. J Allergy Clin Immunol 59:377-382, May 1977

Hamburger RN: IgE and allergy. Adv Pediat 23:95-112, 1976

Occupational Asthma

OCCUPATIONAL ASTHMA refers to a disease in those persons in whom asthma develops primarily from exposures at their work or hobby sites. Although occupational asthma dates at least to Ramazzini's reports in 1700 of asthma provoked by flour dust in bakers and wig makers, the sources of exposures seem to be on the increase in recent years. The provoking agents may be divided into those of (1) *animal origin* affecting poultry farmers; veterinarians; horse, cattle and sheep breeders; chicken inspectors; furriers, and aviarists; (2) *vegetable origin:* farmers, hempworkers, grainworkers, bakers, millers and woodworkers; (3) *inorganic chemicals:* chromium, platinum, aluminum and nickel factory workers, electricians, welders and solderers, and (4) *organic chemicals:* plastic makers and painters who handle isocyanates; meat wrappers exposed to polyvinyl fumes; workers in pharmaceutical plants, and workers using enzymes such as trypsin, Bacillus subtilis enzymes and papain. These are but a few examples of an almost endless list of occupations and causative agents in occupational asthma.

Asthma may occur not only in those who are predisposed by being atopic but also in apparently normal persons without a personal or family history of allergies or IgE specific antibodies. There is some evidence that the atopic persons have a more rapid onset of symptoms following exposures and tend to leave their jobs sooner. Asthma may occur within minutes of a sufficient exposure and responds well to bronchodilators. Others may have asthma delayed several hours after exposure and are better controlled by steroids than by epinephrine or theophylline. Some experience both immediate and late onset asthma.

The pathogenic mechanisms are not always clear-cut. Some agents such as animal danders and enzymes are allergens producing specific IgE antibodies even in nonatopic persons. Others such as polyvinyl and acetylene fumes are respiratory irritants, while still others like toluene diisocyanate (TDI) may have both antigenic and irritant properties.

The examining physician can be alerted to the relationship of asthma to job or hobby by obtaining a careful history. A tip-off is the waxing and waning of symptoms, worse during workdays and better on holidays, weekends and vacations. Sometimes the exposure source may not be readily apparent, such as the case of a secretary whose office is clean but who shares access or ventilation with an adjoining factory. Confirmation that the suspected agent does provoke asthma may require bronchial challenge and immunological studies on the patient and control subjects. Discovery of occupationally induced asthma is rewarding, since removal from the exposure may well effect a cure.

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REFERENCES

Murphy RLH, Jr: Industrial diseases with asthma, *In* Weiss EB, Segal MS (Eds): Bronchial Asthma. Boston, Little, Brown & Co., 1977, p. 517-536

Pepys J: Clinical and therapeutic significance of patterns of allergic reactions of the lungs to extrinsic agents. Am Rev Respir Dis 116:573-588, Oct 1977

Emergency Room Treatment of the Adult Asthmatic

DURING THE COURSE of chronic asthma, acute attacks occur that can be effectively treated in a hospital emergency room. A brief initial history can determine the possible precipitating cause of the attack, such as exposure to allergens or respiratory irritants, intercurrent viral respiratory infections, aspirin or other analgesics, emotional trauma, inadequate maintenance therapy, abrupt withdrawal of steroid medications, or overuse of aerosol bronchodilators.

Severe bronchial obstruction produces decreased breath sounds, notably prolonged expiration, use of accessory muscles of respiration and pulsus paradoxus. Tachycardia and mild hypertension are characteristic of acute asthma and not necessarily complications of treatment. Cyanosis, quiet chest, restlessness and exhaustion are ominous signs of impending or actual respiratory failure

Arterial blood gas measurements are essential